







## ORIGINAL ARTICLE

# Acute onset movement disorders in diabetes mellitus: A clinical series of 59 patients

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## Abstract

**Background and purpose:** No previous study has assessed the frequency and clinical-radiological characteristics of patients with diabetes mellitus (DM) and acute onset non-choreic and nonballistic movements. We conducted a prospective study to investigate the spectrum of acute onset movement disorders in DM.

**Methods:** We recruited all the patients with acute onset movement disorders and hyperglycemia who attended the wards of three hospitals in West Bengal, India from August 2014 to July 2021.

**Results:** Among the 59 patients (mean age =  $55.4 \pm 14.3$  years, 52.5% men) who were included, 41 (69.5%) had choreic or ballistic movements, and 18 (30.5%) had nonchoreic and nonballistic movements. Ballism was the most common movement disorder ( $n = 18$ , 30.5%), followed by pure chorea ( $n = 15$ , 25.4%), choreoathetosis ( $n = 8$ , 13.6%), tremor ( $n = 5$ , 8.5%), hemifacial spasm ( $n = 3$ , 5.1%), parkinsonism ( $n = 3$ , 5.1%), myoclonus ( $n = 3$ , 5.1%), dystonia ( $n = 2$ , 3.4%), and restless leg syndrome ( $n = 2$ , 3.4%). The mean duration of DM was  $9.8 \pm 11.4$  years (89.8% of the patients had type 2 DM). Nonketotic hyperglycemia was frequently (76.3%) detected. The majority (55.9%) had no magnetic resonance imaging (MRI) changes; the remaining showed striatal hyperintensity. Eight patients with MRI changes exhibited discordance with sidedness of movements. Most of the patients (76.3%) recovered completely.

**Conclusions:** This is the largest clinical series depicting the clinical-radiological spectrum of acute onset movement disorders in DM. Of note was that almost one third of patients had nonchoreic and nonballistic movements. Our findings highlight the importance of a capillary blood glucose measurement in patients with acute or subacute onset movement disorders, irrespective of their past glycemic status.

## KEYWORDS

Choreoballism, Diabetes mellitus, Diabetic striatopathy, Hyperglycemia, Movement disorders

Subhankar Chatterjee and Ritwik Ghosh contributed equally and are regarded as joint second authors.

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## INTRODUCTION

Diabetic striatopathy, coined as an umbrella term, is defined as a hyperglycemic condition associated with both or either one of the two following conditions: (i) acute onset chorea-ballism and (ii) striatal hyperdensity on computed tomography (CT) or striatal hyperintensity on T1-weighted magnetic resonance imaging (MRI) [1-12]. Previous clinical series and systematic review regarding acute onset movement disorders in diabetes mellitus (DM) included only patients with chorea or ballism (Table 1) [1-10]. However, in the past few years, different case reports describing other movement disorders apart from chorea and ballism have been reported [13-16]. There is a knowledge gap, as no previous study has assessed the frequency and clinical-radiological characteristics of patients with DM and acute onset movement disorders, including nonchoreic and nonballistic movements. A study on this topic would advance our knowledge of these conditions.

We conducted a prospective study to investigate the spectrum of acute onset movement disorders associated with DM in patients attending the wards of three Indian hospitals. Data on demographics, numerous comorbidities, laboratory values, neuroimaging, outcomes, and treatment were analyzed. This study, comprising 59 patients, is the largest observational study depicting the clinical-radiological spectrum of acute onset movement disorders associated with DM.

## METHODS

All patients with acute onset movement disorders who attended the wards of the Burdwan Medical College and Hospital (Neurology Superspecialty and Internal Medicine Wings), Bangur Institute of Neurosciences, and Berhampore Mental Hospital, in West Bengal, India, between 1 August 2014 and 1 July 2021, were screened with random capillary blood glucose, followed by confirmation with fasting blood glucose, postprandial blood glucose, and glycated hemoglobin according to the guidelines of the American Diabetes Association [17].

History of head injury, visual disturbances, altered sensorium, drowsiness, headache, convulsions, fever, addiction, DM and complicating comorbidities, similar illnesses in family members, atherosclerotic cardiovascular disease, seizures, and drug therapy (current prescription medications) was obtained.

A thorough neurological examination (higher cognitive functions, cranial nerves, fundoscopy, motor, sensory, and autonomic systems, reflexes, cerebellum, meningeal signs, and gait) and a review of other systems were performed. The semiology of the involuntary movements (evaluated in resting state and postural and action-induced whenever applicable) was also noted and classified accordingly as choreic or ballistic (pure chorea, choreoathetosis, and ballism), and nonchoreic and nonballistic movements (myoclonus, dystonia, hemifacial spasm, restless leg syndrome, tremor, and parkinsonism)

**TABLE 1** Previous selected clinical series and systematic reviews of acute onset movement disorders in diabetes mellitus

Authors	Study type	Sample size	Type of movement disorder	Ketosis/nonketosis	Mean age, years
Chua et al. [1]	Systematic review	176	Chorea-ballism	Both	67.6
Ryan et al. [2]	Retrospective study	7	Chorea-ballism	Not mentioned	80
Gómez-Ochoa et al. [3]	Meta-analysis	286	Chorea-ballism	Both	72 (median)
Cosentino et al. [4]	Case series	20	Chorea-ballism	Nonketosis	67.8
Guo et al. [5]	Retrospective study	12	Hemichorea	Nonketosis	74.9
Chen et al. [6]	Systematic review	15	Chorea-ballism	Ketosis	54.1
Prabhu and Ramya [7]	Retrospective study	11	Chorea-ballism-athetosis	Not mentioned	58.7
Lee et al. [8]	Retrospective study	25	Chorea-ballism	Both	73.5
Oh et al. [9]	Meta-analysis	53	Chorea	-	71.1
Lee et al. [10]	Case series	8	Chorea-ballism	-	70

by consensus of two board-certified neurologists (with minimum 5 years of experience in neurology postcertification) [18,19].

A detailed laboratory examination (complete blood cell count, serum sodium, potassium, calcium, magnesium, creatinine, blood urea, blood osmotic concentration, liver function tests, lipid profile, capillary blood glucose, fasting blood glucose, postprandial blood glucose, glycated hemoglobin, thyroid function tests, human immunodeficiency virus 1 and 2, syphilis, hepatitis B and C serologies, urinary albumin-creatinine ratio, antiphospholipid antibodies, and antistreptolysin O antibodies) was performed.

Brain CT scan, brain MRI, magnetic resonance angiography (acquired with a Siemens Verio 1.5-T MRI scanner), neuroelectrophysiological tests (electroencephalography and nerve conduction studies were done in all cases, and electromyography where indicated), chest X-ray, electrocardiography, and two-dimensional and Doppler echocardiography were performed in all patients. In neuroimaging, diffusion-weighted and susceptibility-weighted imaging sequences were analyzed in every case. Striatal changes were mainly considered, whereas other nonspecific incidental findings were omitted for this study. Striatopathy on imaging was further subdivided into two groups, namely, concordant with the clinical presentations (bilateral involuntary movements with bilateral striatopathy or unilateral involuntary movements with contralateral striatopathy) and discordant with the clinical presentations (bilateral involuntary movements with unilateral

striatopathy or unilateral involuntary movements with bilateral striatopathy or unilateral involuntary movements with ipsilateral striatopathy).

Patients with seizures or those with stroke, demyelination, structural intracranial lesion (other than striatopathy), pregnancy, major metabolic perturbation not related to hyperglycemia, history of pre-existing movement disorders, and any other pre-existing neurological disorders were excluded (Figure 1).

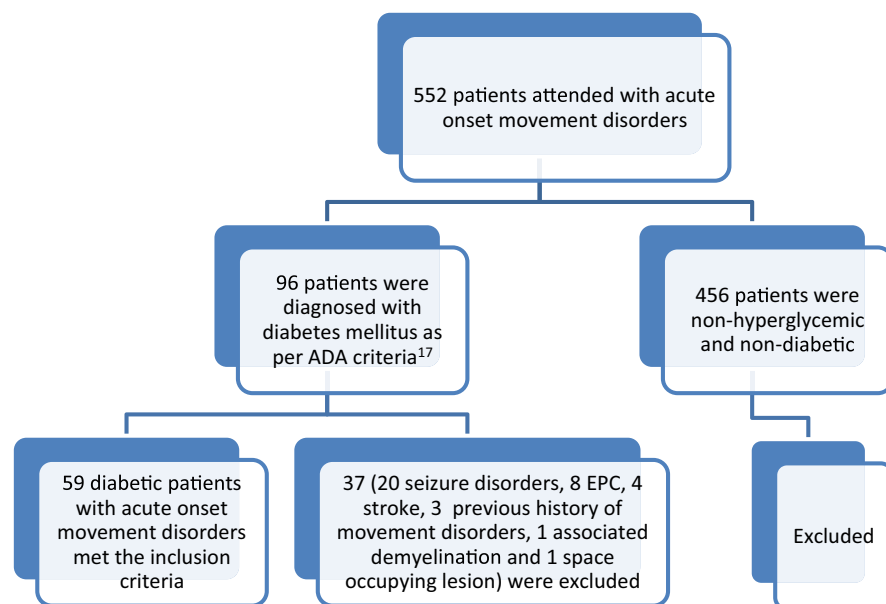
Patients with movement disorders were documented and followed up for at least 3 weeks. Those with complete resolution of movement disorders were assigned to good recovery, whereas those with persisting movements were considered incomplete recovery. Fully recovered patients who were free from all movement disorders within 7 days were regarded as early responders, whereas those who recovered after 7 days were considered late responders.

The institutional ethics committee of the Burdwan Medical College and Hospital, Burdwan, West Bengal, India, approved all procedures on human experimentation. Written (signed) informed consent was obtained from all participants upon enrollment.

## Statistical analyses

Statistical calculations were performed with SPSS Statistics version 21 software (IBM). A  $p$ -value < 0.05 was considered statistically

Men:women	Mean blood glucose, mg/dl	Mean glycated hemoglobin, %	Anatomical area involved	Outcome
1:1.7	414	13.1	Putamen	Successful treatment with glucose control only and neuroleptics. The recurrence rate was 18.2%. There were discrepancies in clinical-radiological manifestations.
1:2.5	Not mentioned	Not mentioned	Putamen	Only one patient had resolution after hyperglycemia correction; all others received neuroleptics.
1:1.7	420 (median)	14 (median)	Putamen	84.9% reported having complete clinical recovery. Neuroleptics were prescribed to 60.8% of the patients.
1:1.3	306.7	Not mentioned	Putamen	All patients recovered. Neuroleptics were prescribed to all patients.
1:3	330.7	11.4	Putamen	Complete clinical resolution in all patients; 41.7% had recurrences; 66.7% needed neuroleptics.
1:2.8	654.4	Not mentioned	Putamen	Complete clinical resolution in 93.3% of the patients; 40% needed neuroleptics.
1:1.8	329.4	11.7	Putamen/caudate	100% clinical recovery. 45.5% received neuroleptics.
1:1.8	451.6	13.5	Not mentioned	A complete clinical resolution was observed in 92%. 76% required neuroleptics.
1:1.8	481.5	14.4	Putamen	73.6% had complete clinical resolution. 13.2% had a recurrence.
All were women	323.5	12.9	Putamen	All had complete clinical recovery. 75% received neuroleptics.



**FIGURE 1** Flowchart showing stratification of total patients attended with acute onset movement disorders in this study. ADA, American Diabetes Association [17]; EPC, epilepsy partialis continua

**TABLE 2** Demographics and diabetes mellitus-related data (N = 59)

Characteristic	Value
Age, years, mean $\pm$ SD	55.4 $\pm$ 14.3
Sex, n (%)	
Male	31 (52.5%)
Female	28 (47.5%)
Diabetes mellitus type, n (%)	
Type 1	6 (10.2%)
Type 2	53 (89.8%)
Diabetes mellitus duration, years, mean $\pm$ SD	9.8 $\pm$ 11.4
Capillary blood glucose upon admission, mg/dl, mean $\pm$ SD	419.6 $\pm$ 89.3
Glycated hemoglobin, %, mean $\pm$ SD	10.5% $\pm$ 1.8%
Time from onset of movement disorders to clinical assessment, days	10.8 $\pm$ 32.9
Chronic diabetic complications, n (%)	
Nephropathy	
Yes	49 (83.1%)
No	10 (16.9%)
Neuropathy	
Yes	30 (50.8%)
No	29 (49.2%)
Retinopathy	
Yes	35 (59.3%)
No	24 (40.7%)
Acute hyperglycemic complications, n (%)	
Ketotic hyperglycemia	14 (23.7%)
Nonketotic hyperglycemia	45 (76.3%)

significant. Continuous variables were reported as the mean (median)  $\pm$  SD and categorical variables as numbers and percentages. Characteristics of the patients were compared using the chi-squared

test or Fisher exact test when appropriate. As the continuous variables were not normally distributed, Mann-Whitney *U* test was used. Because of the large number of statistical tests performed, we used the Benjamini-Hochberg procedure with a defined false discovery rate of 5% [20].

## RESULTS

Of the 552 patients with acute onset movement disorders who attended the wards during the recruitment, 96 patients were diagnosed as diabetic (Figure 1). Of those 96 diabetic patients, 29 were excluded, as they had coexisting other pathologies (20 with seizure disorders, four with stroke, three with a previous history of movement disorders, one with associated demyelination, and one with a space-occupying lesion). In addition, eight patients with epilepsy partialis continua were also excluded from the present study, as it is not categorized as a movement disorder conventionally; rather, it is categorized as a type of simple focal motor status epilepticus in which frequent repetitive muscle jerks, usually arrhythmic, continue over prolonged periods [21]. Moreover, these patients with epilepsy partialis continua had electroencephalographic changes. That is why they were excluded from the study.

Therefore, 59 diabetic patients with acute movement disorders were finally included in the study (Table 2). Thirty-one (52.5%) were men, and 28 (47.5%) were women. The mean age of the selected population was 55.4  $\pm$  14.3 (range = 13–78) years. Among 59 cases, only three patients were diagnosed with DM for the first time when they presented with acute onset movements disorders in the hospital wards. The mean disease duration was 9.8  $\pm$  11.4 years, with 53 (89.8%) of the patients having type 2 DM. Patients presented to the wards after 10.8  $\pm$  32.9 (range = 1–240) days since the onset of movements disorders. Upon admission, mean capillary blood glucose and glycated hemoglobin were 419.6  $\pm$  89.3 mg/dl and

10.5 ± 1.8%, respectively. Among the chronic microvascular complications, retinopathy, neuropathy, and nephropathy were present in 59.3%, 50.8%, and 83.1% of patients. Nonketotic hyperglycemia was frequently (76.3%) detected, whereas ketonuria, as detected on urinalysis, was present in 23.7% of patients.

Table 3 shows the clinical and neuroradiological characteristics of acute movement disorders among diabetic patients. Among the 59 patients included in the study, 41 (69.5%) had choreic or ballistic movements and 18 (30.5%) had nonchoreic and nonballistic movements. Ballism was the most common movement disorder ( $n = 18$ , 30.5%), followed by pure chorea ( $n = 15$ , 25.4%), choreoathetosis ( $n = 8$ , 13.6%), tremor ( $n = 5$ , 8.5%), hemifacial spasm ( $n = 3$ , 5.1%), parkinsonism ( $n = 3$ , 5.1%), myoclonus ( $n = 3$ , 5.1%), dystonia ( $n = 2$ , 3.4%), and restless leg syndrome ( $n = 2$ , 3.4%).

Thirty-seven (62.7%) patients had a unilateral movement disorder, whereas 22 (37.3%) had bilateral clinical. Of note was that 33 (55.9%) of 59 patients had no MRI changes. Of the remaining 26

(44.1%) cases showing MRI changes, the most commonly afflicted regions were putamen and caudate (20/26) and isolated putamen (6/26). Bilateral striatopathy was observed in nine of 26 cases. Importantly, eight (30.8%) of the 26 patients with MRI changes exhibited discordance with sidedness of movements (Table 3).

Thirty-two (54.2%) of the patients needed additional drugs, such as haloperidol or tetrabenazine for ballism and chorea (22 patients), trihexyphenidyl and propranolol for tremor (one patient), levodopa, and carbidopa for parkinsonism (two patients), trihexyphenidyl and clonazepam for dystonia (two patients), carbamazepine for hemifacial spasm (three patients), and clonazepam and pramipexole for restless leg syndrome (two patients), apart from to insulin, to control the movement disorders. The majority ( $n = 28$ , 47.5%) of the patients had early and complete resolution of symptoms, 17 (28.8%) responded late but had a complete reversal, and 14 (23.7%) patients had only partial recovery (Table 3).

While comparing patients with choreic or ballistic movements to those with nonchoreic and nonballistic movements, no statistically significant differences were observed concerning demographics and DM-related and movement disorders-related variables, except for the time (in days) from the onset of movement disorders to the clinical assessment, which was significantly longer in those with nonchoreic and nonballistic movements ( $p = 0.03$ ; Table 4). These differences might be because choreic and ballistic movements are more evident and worrisome for the patients and their family members than nonchoreic and nonballistic movements. In addition, the proportion of bilateral movement disorders was higher in the nonchoreic and nonballistic movement group than in the choreic and ballistic movement group ( $p = 0.001$ ; Table 4).

**TABLE 3** Clinical and neuroradiological characteristics of acute movement disorders among diabetic patients (N = 59)

Characteristic	n (%)
Type of movement disorder	
Choreic and ballistic	41 (69.5% of the total)
<i>Ballism</i>	18 (30.5%)
<i>Pure chorea</i>	15 (25.4%)
<i>Choreoathetosis</i>	8 (13.6%)
Nonchoreic and nonballistic	18 (30.5% of the total)
<i>Tremor</i>	5 (8.5%)
<i>Hemifacial spasm</i>	3 (5.1%)
<i>Parkinsonism</i>	3 (5.1%)
<i>Myoclonus</i>	3 (5.1%)
<i>Dystonia</i>	2 (3.4%)
<i>Restless leg syndrome</i>	2 (3.4%)
Sidedness of involuntary movement	
<i>Unilateral</i>	37 (62.7%)
<i>Bilateral</i>	22 (37.3%)
Brain magnetic resonance imaging changes	
<i>Present</i>	26 (44.1% of the total)
Concordant with clinical presentations	18 (69.2%)
Discordant with clinical presentations	8 (30.8%)
<i>Absent</i>	33 (55.9% of the total)
Drug(s) given other than insulin	
<i>Yes</i>	32 (54.2%)
<i>No</i>	27 (45.8%)
Clinical outcome	
<i>Good and early recovery</i>	28 (47.5%)
<i>Good and late recovery</i>	17 (28.8%)
<i>Poor/incomplete recovery</i>	14 (23.7%)

## DISCUSSION

Unlike previous clinical series and systematic reviews (Table 1) [1-10], the current study also included patients with other movement disorders. Interestingly, almost one third of movement disorders were nonchoreic and nonballistic, which is not the common perception. This underpins the importance of measuring capillary blood glucose immediately with any acute onset movement disorders, including those not classically described in diabetic striatopathy [13-16].

In a smaller case series from South India comprising 11 cases of diabetic chorea-ballism, the mean age was 58.7 years [7], a value much closer to the current study. Compared to most previous studies [1,3,6,8,10], our sample was younger. Whether ethnicity plays any role in this needs further exploration.

A clear predominance of women among patients with hyperglycemic choreic and ballistic movements has been observed across different studies (see Table 1) [1-10]. The exact role of biological sex on hyperglycemia-induced acute movement disorders needs further exploration. Increased sensitivity of the dopamine receptors in the striatum among estrogen-deficient postmenopausal women has been postulated as a possible explanation [6,8]. The slight predominance of men in the current study in contrast with the global scenario might

**TABLE 4** Diabetes mellitus- and movement disorders-related characteristics of the patients upon admission: comparisons of choreic and ballistic movements group versus nonchoreic and nonballistic movements group

Characteristic	Choreic and ballistic movements group, <i>n</i> = 41	Nonchoreic and nonballistic movements group, <i>n</i> = 18	<i>p</i>
Age, years	57.6 (60.0) ± 11.7	50.2 (52.5) ± 18.4	0.229 <sup>a</sup>
Sex, men	22 (53.7%)	9 (50.0%)	0.796 <sup>b</sup>
Duration of diabetes mellitus, years	10.8 (8.0) ± 13.3	7.6 (7.0) ± 4.7	0.547 <sup>a</sup>
Diabetes mellitus type 2	39 (95.1%)	14 (77.8%)	0.064 <sup>b</sup>
Chronic hyperglycemic complications			
Retinopathy	26 (63.4%)	9 (50.0%)	0.334 <sup>b</sup>
Neuropathy	22 (53.7%)	8 (44.4%)	0.514 <sup>b</sup>
Nephropathy	34 (82.9%)	14 (77.8%)	0.721 <sup>b</sup>
Time from onset of movement disorders to clinical assessment, days	5.0 (2.0) ± 11.0	24.1 (6.0) ± 56.1	0.03 <sup>a,c</sup>
Acute hyperglycemic complications			
Ketotic hyperglycemia	10 (24.4%)	4 (22.2%)	1.0 <sup>b</sup>
Nonketotic hyperglycemia	31 (75.6%)	14 (77.8%)	
Sidedness of movement			
Unilateral	33 (80.5%)	4 (22.2%)	0.001 <sup>b,c</sup>
Bilateral	8 (19.5%)	14 (77.8%)	
Drug(s) given other than insulin	22 (53.7%)	10 (55.6%)	0.893 <sup>b</sup>
Clinical outcome			
Good and early recovery	18 (43.9%)	10 (55.6%)	0.679 <sup>b</sup>
Good and late recovery	13 (31.7%)	4 (22.2%)	
Poor/incomplete recovery	10 (24.4%)	4 (22.2%)	
Glycated hemoglobin, %	10.4 (10.3) ± 1.6	10.8 (10.0) ± 2.3	0.889 <sup>a</sup>
Capillary blood glucose, mg/dl	413.6 (402.0) ± 85.0	433.4 (442.5) ± 99.1	0.553 <sup>a</sup>

Note: Mean (median) ± SD and frequency (%) are reported. All significant differences have been corrected for familywise error rate with the Benjamini–Hochberg procedure, with a defined false discovery (FDR) rate of 5%.

<sup>a</sup>Mann–Whitney *U* test.

<sup>b</sup>Chi-squared test or Fisher exact test, when appropriate.

<sup>c</sup>FDR-adjusted *p* < 0.05.

be indicative of gender bias at tertiary health care facilities and pre-  
vailing sex inequality in access to optimum health care [22].

Similar to previous studies [1], the vast majority (*n* = 53) of the patients in this study were diagnosed with type 2 DM. We observed high capillary blood glucose (mean 419.6 mg/dl) at presentation, suggestive of extremely poor recent control of glyce-  
mic status, a finding similar to other studies [1,3]. In our series, the mean duration of DM was 9.8 years, with three cases (5.1%) in whom movement disorders heralded previously undiagnosed DM. In contrast, Chua et al. [1] found that 17% of all reported diabetic striatopathy cases had previously undiagnosed DM. This discrepancy could be due to publication bias, as cases with a rare presentation of a common disease tend to be readily published. Importantly, in our study, 48 (83.1%) patients had associated stig-  
mata of diabetic microangiopathy (retinopathy, neuropathy, and nephropathy), suggesting long-standing poorly controlled DM [14,23,24]. However, acute disturbance of glycemic status may play a more important and consistent role than glycemic durability in movement disorder generation [25].

The majority of patients (76.3%) were negative for ketone bodies. In the setting of nonketotic hyperglycemia, brain metabolism shifts to the alternative anaerobic pathway in the Krebs cycle, resulting in depletion of  $\gamma$ -aminobutyric acid (GABA), an inhibitory neurotrans-  
mitter [1,9,26]. This leads to attenuated inhibition of the thalamus by the medial globus pallidus, resulting in choreic movements in di-  
abetic patients [1,9,26]. On the other hand, GABA can readily be re-  
synthesized from acetoacetate in the ketotic milieu [1,12,14]. Hence, in the ketotic state, GABA level does not usually decrease too low to cause hyperkinetic movements unless other mechanisms, such as cerebrovascular insufficiency or ultrastructural changes in basal ganglia, are present [1,12,14].

Movement disorders were predominantly unilateral (62.7%), which agrees with previous studies [1-5,7,8,9,10,23,26]. It is still an enigma why a systemic metabolic disturbance, like hyperglycemia, leads to a unilateral neurological phenomenon.

Considering the higher sensitivity of MRI compared to CT scan to detect diabetic striatopathy (95.3% vs. 78.9%) [1] MRI was taken as the neuroimaging modality of choice in our study. However, we found



that only 44.1% of cases had disturbances on brain MRI. Previous studies focusing on choreoballistic movements showed that neuroimaging findings suggestive of diabetic striatopathy were evident among 50%–95% of patients [1,3–6,8]. This wide range of variability in the literature could be due to varied use of MRI or CT and nonhomogeneous neuroradiological definition of diabetic striatopathy [1]. In this study, most patients (55.9%) were devoid of any neuroradiological lesion. These data support that the presence of acute onset movement disorders with concurrent hyperglycemia warrants urgent management without waiting for neuroimaging [8]. The lagging of neuroradiological changes behind the clinical manifestations could be due to hyperglycemia-induced ultrastructural changes that occur much earlier than frank structural changes appreciable on MRI [1]. As in previous studies [1–5,7,8,9,10,23,26], our results confirm that, if neuroimaging is abnormal, putamen with or without caudate involvement was the most commonly affected area. Isolated caudate nucleus, globus pallidus, and rarely, subthalamic nucleus involvement have also been previously reported [1–5,7,8,9,10,23,26]; in our series, we did not observe such changes. It is unknown why putamen is particularly vulnerable to hyperglycemia and how the same putaminal lesion leads to wide arrays of different kinds of movement disorders.

Discordance between the neuroradiological lesion and clinical presentation can manifest as bilateral neuroradiological lesions with unilateral clinical presentation, unilateral neuroradiological lesion and bilateral clinical presentations, and same-sided radiological lesion and clinical presentation. There are also reports of frank neuroradiological (striatal) lesions without any clinical (movement disorders) manifestations [27–29], which raises the question of the utility of doing neuroimaging for screening purposes among all poorly controlled diabetics. In our study, 30.8% of cases with neuroimaging changes showed such discordances. Similar findings have been reported by Gómez-Ochoa et al. [3].

The underlying cause of clinical–radiological discordance, which defies the classical concept of neurological localization, needs further study and may be better studied with newer neuroimaging modalities, such as functional MRI [30]. This discordance may be a consequence of widespread involvement of brain circuitries and affliction at ultrastructural levels of neural substrates apart from what is observed in conventional neuroimaging [30]. Nevertheless, this might also be because neural substrates involvement and clinical manifestations in metabolic disturbances are significantly different from hereditary degenerative or vascular disorders. However, this requires further dedicated studies.

In our study, 45.8% of patients had successfully recovered with insulin therapy alone. Lee et al. [10] Guo et al. [5] and Chua et al. [1] reported the use of only hypoglycemic therapy in 24%, 25%, and 25.6% of patients, respectively. In the case series by Cosentino et al., [4] patients needed haloperidol to decrease the abnormal movements. It could be due to the late presentation of the patients after the onset of movement disorders (mean = 33 days) [4]. By contrast, in Prabhu and Ramya's series [7], patients presented much earlier (mean = 2.3 days), resulting in full recovery from movement disorders in the majority (54.5%) of them. Chen et al. [6] reported

even higher recovery (60%) with insulin therapy alone, although they analyzed only ketotic hyperglycemia cases. Additional antichorea drugs used are haloperidol, tetrabenazine, and clonazepam [1].

The recovery rate of the previous studies varied from 76.4% to 100% [1,3,5–10]. This variation could be due to differences in the definition of recovery (clinical and/or neuroradiological) and duration of follow-up. We could only assess the clinical recovery with a follow-up of 3 weeks in the current study. This study is limited by lack of long-term follow-up to assess clinical recurrence or neuroradiological resolution.

In closing, clinicians should know that acute movement disorders might be a DM complication. Our findings highlight the importance of a capillary blood glucose measurement in patients with acute or subacute onset movement disorders, irrespective of their past glycemic status. Ballism followed by pure chorea were the commonest movement disorders identified, although other simple and complex movement disorders may stem from poor glycemic control overall or acute disturbance of blood glucose level. Clinical–radiological discordance is not rare. Treatment with insulin alone helps alleviate abnormal movements, although the role of adding on other medications cannot be ignored.

## CONFLICT OF INTEREST

None of the authors has any conflict of interest to disclose.

## AUTHOR CONTRIBUTIONS

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## DATA AVAILABILITY STATEMENT

The data used to support the findings of this study are included in the article.

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